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## **COMMON SERUM CHEMISTRIES**

<b>Albumin</b>	<b>3.5-4.8 gm/dl</b>
<b>Bicarbonate [HCO<sub>3</sub><sup>-</sup>]</b>	<b>23-28 mEq/L</b>
<b>Blood gases (arterial, whole blood)</b>	
<b>pH</b>	<b>7.35-7.45</b>
<b>PO<sub>2</sub></b>	<b>80-105 mm Hg</b>
<b>PCO<sub>2</sub></b>	<b>34-45 mm Hg</b>
<b>Calcium</b>	
<b>Total</b>	<b>9.0-10.3 mg/dl</b>
<b>Free</b>	<b>4.5-5.0 mg/dl</b>
<b>Carbon Dioxide content (bicarbonate)</b>	<b>24-32 mEq/L</b>
<b>Chloride</b>	<b>95-105 mEq/L</b>
<b>Cholesterol</b>	<b>1-239 mg/dl</b>
<b>Creatinine</b>	
<b>Male</b>	<b>0.8-1.3 mg/dl</b>
<b>Female</b>	<b>0.6-1.1 mg/dl</b>
<b>Glucose (fasting, plasma)</b>	<b>65-110 mg/dl</b>
<b>Magnesium</b>	<b>1.7 – 2.7 mg/dL (1.4 –2.3 mEq/L)</b>
<b>Osmolality</b>	<b>280-290 mOsm/kg water</b>
<b>Phosphorus (inorganic)</b>	<b>2.5-4.5 mg/dl</b>
<b>Potassium</b>	<b>3.5-4.5 mEq/L</b>
<b>Protein (total)</b>	<b>6.5-8.5 gm/dl</b>
<b>Sodium</b>	<b>135-145 mEq/L</b>
<b>Urea nitrogen (BUN)</b>	<b>8-25 mg/dl</b>

## CASES I and II

You are working in the emergency department when two patients are brought in by ambulance both short of breath.

Patient A is a 60 year old male with a history of two previous myocardial infarctions. He notes that for the last week he has become progressively more short of breath. He is unable to walk up a flight of stairs without resting. He sleeps on three pillows at night and for the last two nights has awoken 2 hours following falling asleep with shortness of breath. He also notes a progressive weight gain of 15 pounds over the last two weeks associated with increased swelling of his legs up to the level of his knees. He denies chest pain, nausea or emesis, diaphoresis, cough or fever.

Past medical history is significant for coronary artery bypass grafting 3 years previously and hypertension for 15 years. He has a 60 pack year history of cigarette use and denies alcohol or illicit drug use.

Medications: Digoxin 0.25 mg qd, hydrochlorothiazide 25 mg qd, atenolol 50 mg qd.  
Family history negative for coronary artery disease, diabetes mellitus, sickle cell anemia.  
Social history: Previously employed as a construction worker, on disability for 3 years.

### Physical exam:

Vital signs: Blood pressure 90/60, pulse 50 regular, respiratory rate 20, afebrile

General appearance: alert, oriented male well nourished appearing stated age

HEENT: sclera anicteric, fundi AV nicking, copper wiring

Neck: no thyromegaly, adenopathy

Cardiac: JVD to angle of mandible at 45 degrees, no RV lift or thrill, PMI laterally displaced with a rocking precordium, carotids decreased upstroke. Auscultatory: soft S1, S3 present.

Lungs: Crackles to mid chest. No wheezing or rhonchi.

Abdomen: liver palpable 3 cm below right costal margin with smooth edge. Positive hepatojugular reflux. No splenomegaly or fluid wave.

Extremities: 3 plus pitting edema to knees. No clubbing or cyanosis. Pulses 1+ and symmetrical, no bruits.

Neurological: Mental status intact, no asterixis.

### Laboratory data:

sodium 122 mEq/L, potassium 3.5 mEq/L, chloride 95 mEq/L, bicarbonate 18 mEq/L, BUN 60 mg/dL, creatinine 2.0 mg/dL, glucose 90 mg/dL

plasma osmolality 269 mEq/kg, urine osmolality 400 mEq/kg, urine sodium 15 mEq/L

Albumin 4.0 gm/dL, total protein 7.5 g/dL, cholesterol 210, triglycerides 190

WBC 6.0, hemoglobin 11 g/dL, hematocrit 38 percent.

Urinalysis: specific gravity 1.025, 1+ protein

Chest X ray: enlarged LV, cephalization of pulmonary vasculature, Kerley B lines, bilateral pleural effusions.

1. How would you characterize the volume status of this patient? What factors in the history, physical and laboratory data support this determination?

2. Is this a hypoosmolar hyponatremia and how did you decide?

3. Is there an osmolar gap?

4. Are the kidneys responding to clear a free water load?

5. Is the patient in a sodium retaining state?

6. What is the basis for the patient's hyponatremia?

7. What other conditions result in hyponatremia in the setting of hypervolemia and how would you distinguish them from this presentation?

8. What drug that the patient is taking may have contributed to the hyponatremia?

9. What hormone(s) are contributing to the patient's volume and electrolyte disturbance?

10. What is the best way to treat the hyponatremia? Should the resolution of the hyponatremia be the primary goal of therapy?

11. Should the use and monitoring of any of his current medications be changed?

Patient B is a 52 year old male. He was in his previous state of health until 3 hours prior to his arrival in the emergency department. At that time he suffered the sudden onset of precordial chest pressure that radiated to his neck and left arm. The pain resolved after 45 minutes. The pain was accompanied by the onset of nausea and emesis. In addition, the patient has been persistently diaphoretic and markedly short of breath. Cardiac risk factors include a 30 pack year history of smoking, hypercholesterolemia, diabetes mellitus for five years and a family history with a father and brother who died in their 30s of a myocardial infarction.

Past medical history is significant for diabetes mellitus.

Medications: atorvastatin 10 mg qd and glyburide 3 mg qd.

Physical exam:

Vital signs: BP 110/80, pulse 100, respiratory rate 24, afebrile

General appearance: Well developed, well nourished male in moderate respiratory distress.

Chest: bibasilar crackles

Cardiovascular: jugular veins non-visualized, RV no lift or thrill, PMI discrete in midclavicular line, carotids normal upstroke and amplitude. Auscultatory findings, tachycardic, regular rhythm, no S3 or rub, 2/6 systolic murmur at base.

Abdomen: nontender, without organomegaly

Extremities: no edema, clubbing or cyanosis.

Laboratory data:

sodium 140 mEq/L, potassium 3.5 mEq/L, chloride 95 mEq/L, BUN 15 mg/dL, creatinine 1.0 mg/dL, glucose 150 mg/dL, urine Na<sup>+</sup> <20

Albumin 4.0 gm/dL, total protein 7.5 g/dL

WBC 12.0, hemoglobin 12 g/dL, hematocrit 42 percent.

Urinalysis: specific gravity 1.025, 1+ glucose

Chest X ray: normal cardiac silhouette, cephalization of pulmonary vasculature, Kerley B lines, patchy alveolar infiltrates

1. How would you characterize the volume status of this patient? What factors in the history, physical and laboratory data support this determination?

2. Knowing the patient's volume status, in what way would your acute treatment of his pulmonary edema differ from patient A? Would diuretics be indicated?

3. If you were to place a Swan-Ganz catheter in both patients A and B how would the chamber pressures and PA occlusive pressures differ?



### CASE III

A 45 year old male is brought to the emergency room because of weakness, nausea, and vomiting. He had been drinking alcohol heavily for a week until 24 hours earlier and has had decreased dietary intake. His blood pressure is 110/70 with a heart rate of 110, respiratory rate of 30, and a temperature of 102 F. Labs sent from the emergency room just before he has a seizure return and show:

$\text{Na}^+ = 137 \text{ mEq/L}$

$\text{K}^+ = 2.8 \text{ mEq/L}$  (low)

$\text{Cl}^- = 86 \text{ mEq/L}$  (low)

$\text{HCO}_3^- = 37 \text{ mEq/L}$  (high)

$\text{pCO}_2 = 33$  (low)

$\text{pH} = 7.67$  (high)

$\text{Mg}^{2+} = 0.8 \text{ mEq/L}$  (low)

$\text{Ca}^{2+} = 7.0 \text{ mg/dL}$  (low)

Albumin = 3.5 mg/dL (normal)

Glucose = 60 mg/dL (low)

1. Multiple derangements contribute to hypokalemia in this case including redistribution, decreased intake, renal, and extra renal losses. For each of these, explain the specific contributory factors based on the history and labs and how they promote the hypokalemia.

2. What will happen to the patient's serum  $K^+$  if we administer 5% dextrose as immediate IV fluid replacement to help correct the hypoglycemia?

3. What acid-base disturbance is present and how did it develop?

4. What would the urine chloride be?

5. What types of fluid and electrolyte replacement will this patient require?

## CASE IV

A 63 year old previously non insulin-dependent diabetic has been experiencing increasing polyuria and polydipsia for several weeks and is found to have a finger stick glucose of 500 at her physician's office. She is sent to the ER. History there reveals that she has been checking her blood pressure at home and it has been running high, so she picked up a salt substitute at Kroger's and has been using it heavily to flavor her food. Her medications include metoprolol (beta blocker), enalapril (angiotensin converting enzyme inhibitor), glipizide (oral hypoglycemic), and ibuprofen as needed for arthritis. Her blood pressure is 180/100, pulse 65. She has poor veins and an M3 draws her blood.

Initial chemistries return:

$\text{Na}^+ = 142 \text{ mEq/L}$

$\text{K}^+ = 7.9 \text{ mEq/L}$  (very high)

$\text{Cl}^- = 105 \text{ mEq/L}$

$\text{HCO}_3^- = 14 \text{ mEq/L}$  (low)

Glucose = 550 mg/dL (high)

BUN = 35 mg/dL (high)

Creatinine = 2.0 mg/dL (high)

The lab indicates that the blood is slightly hemolyzed.

1. What is contributing to the hyperkalemia here?

2. The M3 asks his intern to redraw the sample. What can they do in the meantime to decide on the urgency and intensity of therapy needed?

3. The additional studies come back positive. How should the patient be treated acutely and what does each of the treatments do?

4. How can this problem be avoided in the future?

5. Is there anything suggesting the presence of an acid-base disturbance?